Allergic Inflammatory Mediated Coronary Artery Vasospasm: A Case Report of Kounis Syndrome

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INTRODUCTION
Kounis syndrome is an acute coronary syndrome occurring secondary to an anaphylactic reaction. A wide variety of triggers such as drugs and environmental exposures have been described for the so-called allergic myocardial infarction. The pathophysiology of Kounis syndrome is thought to be due to the release of inflammatory mediators such as histamine and cytokines by mast cells and eosinophils resulting in either coronary vasospasm, myocardial infarction or stent thrombosis.

CASE PRESENTATION
A 57 year-old African American woman was brought in by ambulance from home with acute onset of chest tightness that awoke her from sleep that morning. The pain was associated with nausea, shortness of breath, and numbness with paresthesias in her extremities. Upon presentation, her physical exam was notable for a diffuse erythematous, non-blanching, maculopapular rash on the trunk as well as the extremities (Figure 1). Laboratory results were significant for an elevated white blood cell count of 19.4 x 10^9/L with 2.4% eosinophils (normal 1-6%), and troponin T < 0.01. An EKG demonstrated 2mm ST segment elevations in leads II, III and aVF concerning for inferior wall ischemia (Figure 2A).

The patient underwent an urgent cardiac catheterization due to concern for acute coronary syndrome. However, cardiac catheterization did not show any occlusion or significant stenosis of the coronary arteries (Figure 3). A repeat EKG was performed following catheterization, which demonstrated resolution of the ST segment elevations (Figure 2B). The absence of coronary artery disease suggested that these transient ST segment elevations seen in the inferior leads of the EKG were due to coronary artery vasospasm rather than coronary plaque rupture or thrombus. A transthoracic echocardiogram obtained following cardiac catheterization showed no wall motion abnormalities with normal ejection fraction.

DIFFERENTIAL DIAGNOSIS
The patient underwent an extensive work-up to determine the cause of her rash. Her infectious and autoimmune workups were unremarkable. Later during her hospitalization, the patient developed an eosinophilia with a white blood cell differential containing 20% eosinophils. A punch biopsy of the skin was performed, which showed superficial perivascular dermatitis with eosinophils and neutrophils pointing to a hypersensitivity reaction. Upon further questioning, it was revealed that the patient was started on mirtazapine by her primary care physician for treatment of insomnia three days prior to admission. Because there was no other identified etiology from her work-up, mirtazapine was suspected to be the culprit of her allergic reaction. Subsequently, mirtazapine was stopped and the patient was given a course of oral glucocorticoids which led to resolution of the rash.
DISCUSSION

Kounis syndrome was originally described in 1991; however, this phenomenon is not widely known in clinical practice. Above we have described a case of Kounis syndrome in which coronary artery vasospasm occurs due to inflammatory mediators in the absence of underlying coronary artery disease. It is clear from her diffuse rash, eosinophilia, and skin biopsy that she was undergoing an allergic inflammatory reaction which ultimately led to her coronary artery vasospasm. Recognizing Kounis syndrome as a differential diagnosis for acute coronary syndrome is important as treatment with steroids and antihistamines may be of benefit in addition to vasodilators such as calcium-channel blockers and nitrates.

REFERENCES